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EXAMINER	
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ART UNIT	PAPER NUMBER
1635	

NOTIFICATION DATE	DELIVERY MODE
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

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Office Action Summary	Application No.	Applicant(s)	
	10/539,527	GIRARD ET AL.	
	Examiner	Art Unit	
	DANA SHIN	1635	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 April 2010 and 27 July 2010*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *23,25,26,28,127 and 130* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *23,25,26,28,127 and 130* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *4-6-2010*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Status of Application/Amendment/Claims

This Office action is in response to the communications filed on April 19, 2010 and July 27, 2010.

Currently, claims 23, 25-26, 28, 127, and 130 are pending and under examination on the merits in the instant case.

The following rejections are either newly applied or are reiterated and are the only rejections and/or objections presently applied to the instant application.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Response to Arguments and Amendments

Withdrawn Rejections

Any rejections not repeated in this Office action are hereby withdrawn.

Maintained Rejections

Claim Rejections - 35 USC § 103

Claims 23 and 25 remain rejected under 35 U.S.C. 103(a) as being unpatentable over King et al. and Lewis et al. for the reasons of record as set forth in the Office action mailed on December 17, 2009 and for the reasons stated below.

Applicant's arguments filed on April 19, 2010 have been fully considered but they are not persuasive. Applicant argues that the claims are not obvious because the combination of King et

al. and Lewis et al. would not have motivated one skilled in the art to consider using an siRNA against NF-HEV for chronic inflammation treatment. Contrary to applicant's argument, the combination of the cited prior art references provides sufficient guidance and motivation to inhibit SEQ ID NO:1879 of King et al. with an siRNA molecule to treat chronic inflammation. Note that contrary to applicant's argument that SEQ ID NO:1879 relates only to cancer treatment methods, SEQ ID NO:1879 was "claimed" to be useful in a method for stimulating T cells (immune cells) specific for a tumor protein. See claim 9. Further, administration of SEQ ID NO:1879 was "claimed" to stimulate an immune response in a patient. See claim 12. Hence, contrary to applicant's biased interpretation of the teachings of King et al., King et al. explicitly disclosed that a polynucleotide SEQ ID NO:1879 encoding human DSV27-related protein is an immunostimulatory agent that can be used in a therapeutic setting to stimulate immune responses in a patient. Hence, it logically flows that a nucleic acid-based agent (antisense oligonucleotides, ribozymes, PNAs; see paragraphs 2018-2035) that inhibits or reduces the immunostimulatory activity of SEQ ID NO:1879 would necessarily function as an agent that inhibits immune responses in a patient, in particular those responses mediated by a T cell. Further, note that it has long been known in the art that T cell stimulation/activation results in the secretion/production of pro-inflammatory cytokines such as IL-2, IL-12, TNF and IFN-gamma. See paragraphs 2094 and 2122. As such, the claimed methods of stimulating T cells or immune responses in a patient comprising administering SEQ ID NO:1879 of King et al. will inherently result in an increased secretion/production of pro-inflammatory cytokines such as IL-2, IL-12, TNF and IFN-gamma in the patient. In addition, Lewis et al. explicitly taught that inhibiting "cytokines could also treat inflammatory diseases such as arthritis." See paragraph 0009. Hence, it logically flows that performing the opposite method step of King et al., that is, administering a nucleic acid-based

inhibitor of SEQ ID NO:1879 thereby deactivating T cell stimulation and reducing immune responses in a patient would necessarily decrease the amount of pro-inflammatory cytokines produced or secreted by the T cells in the treated patient, which will thus inhibit inflammation in the patient as claimed in the instant case. Now, although King et al. disclosed using antisense oligonucleotides, ribozymes, or PNAs against SEQ ID NO:1879 for inhibition of SEQ ID NO:1879, they did not teach using an "siRNA" against SEQ ID NO:1879 for reducing pro-inflammatory or immune responses in T cells of a patient. However, at the time of filing, using a target sequence-specific siRNA was one of art-recognized target inhibition methods including inhibiting immune responses in a mammal, for example, in a method for treating inflammatory diseases such as arthritis as taught by Lewis et al. Hence, the combined teachings of the cited prior art references and the state of the art/technology and the level of knowledge disclosed by the cited prior art references would not reasonably motivated one skilled in the art to make and use an siRNA against SEQ ID NO:1879 to inhibit inflammation in a patient such as an arthritis patient by reducing pro-inflammatory cytokines produced by stimulated T cells.

Applicant, for some reason, mentions "Kasuya" reference to argue for the asserted nonobviousness of the claims. Applicant argues that the function of DVS27 was not indicated by "Kasuya" because the expression of DVS27 was taught to change in response to unidentified inflammatory stimuli. Applicant's arguments addressing the "Kasuya" reference are irrelevant to the instant ground of rejection and/or how the claims would not have been obvious in view of the combined teachings of King et al. and Lewis et al. Further, contrary to applicant's argument that the function of DVS27 was not clearly suggested by Kasuya et al., Kasuya et al. explicitly suggested that DVS27 encodes a nuclear protein involved in inflammatory responses and is highly up-regulated in response to inflammatory stimuli. Hence, it was reasonably suggested that

DVS27 mediates inflammatory responses in a cell. It appears that applicant is trying to argue for unexpected results as applicant has pointed out paragraphs 0038, 0055, 00382, 0449, and 0452 of the instant specification to assert that the instant specification is the "first" disclosure in the art that teaches reducing NF-HEV ameliorates inflammation. In so arguing, applicant states that the specification shows several pro-inflammatory chemokines such as CCL2/MCP1 are decreased when NF-HEV level/activity is reduced. Contrary to applicant's assertion that the instant specification is the "first" to suggest such relationship between NF-HEV and pro-inflammatory cytokines, the combination of King et al. and Lewis et al. as well as Kasuya et al. suggested the functional role of a gene encoding NF-HEV in promoting production or pro-inflammatory cytokines, thereby increasing inflammation or immune responses in a subject. Further, it appears that applicant is trying to assert the nonobviousness of the claims by differentiating the "unidentified" inflammatory stimuli of Kasuya et al. as opposed to CCL2/MCP1 disclosed in the instant specification. In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., reducing NF-HEV reduces CCL2/MCP1) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

Since applicant's arguments are not persuasive, this rejection is maintained.

Claims 23, 25, and 127 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Lipman, Woolf et al., and Elbashir et al. for the reasons of record as set forth in the Office action mailed on December 17, 2009 and for the reasons stated below.

Applicant's arguments filed on April 19, 2010 have been fully considered but they are not persuasive. Applicant argues that the claims are not obvious because there is no evidence that a reduction in pain will reduce inflammation or vice versa. Applicant's attention is directed to the fact that non-steroidal anti-inflammatory drug (NSAID) was explicitly taught to be useful for treating a chronic inflammation condition, osteoarthritis, which is often accompanied by chronic pain in the joints, which is the major "symptom" associated with osteoarthritis. See Lipman. Note that the claims recite "identifying a subject having symptoms of a condition associated with chronic inflammation" and "inhibiting inflammation" in the subject. At the time of filing, NSAID useful for treating osteoarthritis and the accompanying pain was also independently taught by Woolf et al. as a pain reducer. Woolf et al. also taught that instead of administering NSAID, one can administer an siRNA targeted to a gene that is overexpressed in a subject having chronic pain, wherein the gene is SEQ ID NO:11450 that comprises SEQ ID NO:1 of the instant application. In other words, Woolf et al. suggested that an inhibitor of SEQ ID NO:11450 is a functional equivalent of NSAID, an anti-inflammatory drug, thereby suggesting that an inhibitor of SEQ ID NO:11450 has an anti-inflammatory activity. Furthermore, Woolf et al. taught that SEQ ID NO:11450 is differentially expressed in "chronic constriction pain model" and an "inflammation pain model". See paragraph 0077. Hence, the combined teachings of Lipman and Woolf et al. do suggest that one can treat the chronic pain associated with a chronic inflammatory disease osteoarthritis with an siRNA that inhibits the inflammatory activity of SEQ ID NO:11450.

Applicant argues that there is no showing that "each of the subset of the 14715 sequences that are over-expressed in pain would in fact predictably result in a reduction in pain if targeted with an siRNA." Contrary to applicant's argument, siRNAs against all of 14715 polynucleotides

disclosed in the Woolf et al. reference is a "finite" number of "identified" and "predictable" solutions to treat chronic pain associated with inflammation because Woolf et al. explicitly disclosed the following in paragraph 0067: "The invention further provides a method of treating pain in an animal comprising administering to the animal a double stranded RNA molecule wherein one of the strands of the double stranded RNA molecule is identical to a portion of an mRNA transcript obtained from one or more of the polynucleotide sequences indicated in Table 1, 2, 3, 4, or 5." As such, Woolf et al. taught that inhibiting each and every single polynucleotide disclosed in the reference by siRNA is equally effective for treating pain in an animal, thereby providing a "finite" number of "identified" and "predictable" solutions to treat chronic pain associated with inflammation. Much better, Woolf et al. "claimed" a pain treatment method with an siRNA targeted to any polynucleotide disclosed in Table 1 or Table 2. See claim 45. Note that SEQ ID NO:11450 is a human nucleotides (AB024518; citation of record) encoding SEQ ID NO:11451 (BAA75892; citation of record), both of which are disclosed in Table 2. Courtesy copy of Table 2 (page 605) is provided herewith. See the attached citation. Note that all human polynucleotide and polypeptide sequences disclosed in Table 2 of Woolf et al. are differentially overexpressed by "at least 1.2 fold across at least three replicates screens of neuronal tissue obtained from an animal subjected to pain relative to an animal no subjected to the same pain, with a P-value of less than 0.05.", wherein the animal is of "nerve injury and inflammation pain models". See paragraphs 0009, 0013, and 0170. Hence, SEQ ID NO:11450 encoding SEQ ID NO:11451 is one of finite number of predictable, identified pro-inflammatory, pro-pain genes suggested to be useful to be targeted by an siRNA molecule for treating pro-inflammation pain in a subject.

Applicant addresses "Kasuya et al." and "Onda et al." and argues that the instant specification is the "first" disclosure in the art that teaches reducing NF-HEV ameliorates inflammation. In so arguing, applicant states that the specification shows several pro-inflammatory chemokines such as CCL2/MCP1 are decreased when NF-HEV level/activity is reduced. Contrary to applicant's assertion that the instant specification is the "first" to suggest that "NF-HEV has pro-inflammatory properties", the combination of Lipman and Woolf et al. suggested SEQ ID NO:11450 as a pro-inflammatory gene as Woolf et al. taught that it is differentially overexpressed in "nerve injury and inflammation pain models" and that an siRNA against SEQ ID NO:11450 is a better substitute for NASID, an anti-inflammatory drug. In Further, it appears that applicant is trying to assert the nonobviousness of the claims by differentiating the "unidentified" inflammatory stimuli of Kasuya et al. as opposed to CCL2/MCP1 disclosed in the instant specification. In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., reducing NF-HEV reduces CCL2/MCP1) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

Since applicant's arguments are not persuasive, this rejection is maintained.

Claims 23, 25-26, 28, and 127 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Onda et al., GenBank accession Nos. AB024518 and BAA75892, Lewis et al. and Elbashir et al. for the reasons of record as set forth in the Office action mailed on December 17, 2009 and for the reasons stated below.

Applicant's arguments filed on April 19, 2010 have been fully considered but they are not persuasive. Applicant argues that the claims are not obvious because Onda et al. stated that the "functional role" of DVS27 was "still unknown". It is true that the Onda et al. reference contains such statement. However, a prior art reference must be considered "as a whole" or in its entirety for its teachings and suggestions. The studies performed by Onda et al. unequivocally teach that DVS27 expression is "most highly upregulated in vasospastic arteries" among previously unknown genes (see page 1287) and that their experimental results are "consistent with these observations that inflammatory reactions are closely related to the development of cerebral vasospasm". See page 1287, left column. Onda et al. also show that DVS27 mRNA expression is highly significantly upregulated in response to IL-1α, IL-1β, IFN-γ and is slightly increased in response to TNF-a compared to a negative control. See Figure 7. As such, the teachings of Onda et al. taken as a whole suggest that DVS27 mediates inflammatory reactions and that DVS27 expression level is highly dependent on pro-inflammatory cytokines.

Applicant argues that Onda et al. do not teach whether DVS27 is pro-inflammatory or anti-inflammatory. Contrary to applicant's argument, the Onda et al. reference taken as a whole "reasonably", if not "absolutely", suggests that DVS27 is highly likely to be a pro-inflammatory gene as it is most highly upregulated in cerebral vasospasm arteries that involve "inflammatory reactions" and that DVS27 mRNA expression level is induced by pro-inflammatory cytokines. Note that for obviousness under §103, "all that is required is a reasonable expectation of success", and it does not require "absolute predictability of success". See *In re O 'Farrell*, 853 F.2d 894, 7 USPQ2d 1673 (Fed. Cir. 1988) at 1681.

Applicant argues that Onda et al. do not teach DVS27 mRNA is induced in high endothelial venule (HEV)-like vessels. Note that no claim requires a specific, physical

localization for NF-HEV to be reduced. That is, the rejected claims do not require that DVS27 be reduced only, exclusively in HEV-like vessels. Note that claim 23 as currently amended requires inhibition of inflammatory response in "endothelial cells" of a subject. Note that total RNA of Onda et al. was extracted from various tissues including middle cerebral artery, and therefore, it was reasonably expected, at the time of filing, that DVS27 is expressed in endothelial cells and that an siRNA targeted to DVS27 is reasonably expected to inhibit inflammation in endothelial cells of a subject as currently claimed. Further, in response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., reducing DVS27 expression in HEV-like vessels) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

Applicant argues that Onda et al. do not teach modulating "chronic inflammation such as that seen in rheumatoid arthritis, Crohn's disease or inflammatory bowel disorder". Note that the claims are drawn to a method of "inhibiting inflammation" in a subject having "symptoms of a condition associated with chronic inflammation". Further, note that the rejected claims do not require the specific conditions. Furthermore, applicant's attention is directed to the fact that the instant rejection is an obviousness rejection, not an anticipation rejection. As stated hereinabove, the Onda et al. reference taken as a whole reasonably suggests that DVS27 expression contributes to the inflammatory reactions observed in canine models of "continuous vasospasm of cerebral arteries after subarachnoid hemorrhage", thereby suggesting the association of DVS27 with symptoms of a condition associated with chronic inflammation as recited in the rejected claims.

Applicant argues that Onda et al. do not teach CCL2/MCP1 disclosed in the instant specification. In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., reducing NF-HEV reduces CCL2/MCP1) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification, limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993). In addition, note that the "objective evidence of nonobviousness must be commensurate in scope with the claims which the evidence is offered to support." (emphasis added). See MPEP 716.02.

Since applicant's arguments are not persuasive, this rejection is maintained.

New Rejections Necessitated by Amendment

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claim 130 is rejected under 35 U.S.C. 103(a) as being unpatentable over van Dieten et al. (*Annals of the Rheumatic Diseases,* 2000, 59:753-759) in view of Woolf et al. (US 2007/0015145 A1, citation of record).

van Dieten et al. teach that that chronic pain is one of the symptoms of rheumatoid arthritis and osteoarthritis, a chronic inflammation condition, and therefore patients having arthritis are recommended to take an anti-inflammatory drug such as a non-steroidal anti-

inflammatory drug (NSAID), which causes adverse side effects such as heartburn, abdominal pain, and dyspepsia, which in turn impose high costs for treatment of such adverse side effects. See the entire reference. van Dieten et al. do not teach using an siRNA targeted to SEQ ID NO:1.

Woolf et al. teach that there is an art-recognized need to identify therapeutic strategies other than non-steroidal anti-inflammatory drugs (NSAIDS) for more effective pain management. Woof et al. teach that one can treat chronic pain in a subject by administering an siRNA targeted to a polynucleotide of SEQ ID NO:11450 that is differentially over-expressed in the subject having chronic pain instead of administering NSAIDS, wherein the polynucleotide of 2645 nucleotides in length of SEQ ID NO:11450 is identical to the entire 2645 nucleotides of SEQ ID NO:1 of the instant application and that SEQ ID NO:11450 encodes SEQ ID NO:11451, whose 270 amino acids are identical to the 270 amino acids of SEQ ID NO:4 of the instant application. They teach that therapeutic siRNA molecules base pair with the endogenous transcripts, thereby inhibiting translation of the target mRNA. They teach that "nerve tissue" comprises endothelial cells. See paragraphs 0003-0005, 0015, 0077-0078, 0081, 0084, 0283-0287, 0376; claims 44-45.

It would have been obvious to one of ordinary skill in the art at the time the invention was made to practice the arthritis treatment method by administering an siRNA targeted to SEQ ID NO:11450, which is identical in sequence to SEQ ID NO:1 claimed in the instant case, in place of NSAID.

One of ordinary skill in the art would have been motivated to do so with a reasonable expectation of success so as to cost-effectively and more safely treat patients with rheumatoid arthritis or osteoarthritis, because NSAID was known to cause many adverse side effects that are costly to treat as taught by van Dieten et al., and because at the time of filing it was known in the

art that instead of administering NSAID, one can administer an siRNA targeted to a gene that is overexpressed in "nerve tissues" comprising endothelial cells in a subject having chronic pain as observed in an animal that is of "nerve injury and inflammation pain models" (see paragraph 0170), wherein the gene is SEQ ID NO:11450 is identical to SEQ ID NO:1 of the instant application. In other words, Woolf et al. suggested that an inhibitor of SEQ ID NO:11450 is a functional equivalent of NSAID, an anti-inflammatory drug, thereby suggesting that an inhibitor of SEQ ID NO:11450 has an anti-inflammatory activity. Furthermore, Woolf et al. taught that SEQ ID NO:11450 is differentially overexpressed in "chronic constriction pain model" and an "inflammation pain model" and "nerve injury and inflammation pain models". See paragraphs 0077 and 0170. Hence, the combined teachings of van Dieten et al. and Woolf et al. do suggest that one can treat chronic joint pain (a symptom) associated with rheumatoid arthritis or osteoarthritis with an siRNA that inhibits the inflammatory activity of SEQ ID NO:11450 in an endothelial cell of a subject having rheumatoid arthritis or osteoarthritis. Accordingly, the claims taken as a whole would have been *prima facie* obvious at the time of filing.

Conclusion

No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after

the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to DANA SHIN whose telephone number is (571)272-8008. The examiner can normally be reached on Monday through Friday, 7am-3:30pm EST.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christopher Low (Acting SPE) can be reached on 571-272-0951. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Dana Shin
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